REMARKS

Amendments to the Claims

Claims 1-15, 17-28, and 26-30 are pending. Claims 2-7, 13-15 and 20-25 have been previously withdraw without prejudice as drawn to a non-elected invention. Claim 16 has been previously canceled without prejudice or disclaimer. New claims 29 and 30 have been added. Claims 1, 8, 11, 17, 18 and 26 have been amended.

Claim 1 has been amended to clarify that SEQ ID NO: 5 is SNF1LK and to clarify that the functionally active fragment of SEQ ID NO: 5 encodes a polypeptide comprising amino acid residues 27 – 278 of SNF1LK. Claim 1 has also been amended to delete steps (d) – (f). Support for the amendment is found throughout the specification, particularly at pages 5 and 36, and in original claim 1. Applicants note that SEQ ID NOs 5 and 15 correspond with the nucleic acid and polypeptide for SNF1LK, respectively (see GenBank # 27597093 and GenBank # 27597094).

Claim 8 have been amended to clarify that the nucleic acid is SNF1LK and that the nucleic acid modulator modulates the expression of SNF1LK.

Claim 11 has been amended merely to provide proper antecedent basis by reciting step (d) rather than step (g).

Claims 17 and 18 have been amended merely to correct their dependencies to depend from new claim 29.

Claims 26 has been amended to clarify that the expressed polypeptide is SEQ ID NO: 15 (SNF1LK) or a polypeptide encoded by a polynucleotide comprising SEQ ID NO: 5 (SNF1LK) and to clarify that the functionally active fragment of SEQ ID NO: 15 or encoded by a polynucleotide comprising SEQ ID NO: 5 comprises amino acid residues 27 – 278 of SNF1LK. Support for the amendment is found throughout the specification and particularly at pages 5 and 36.

New claim 29 recites "[t]he method of claim 1, comprising the additional steps of: (e) providing a second assay system comprising cultured cells or a non-human animal expressing MARK, wherein the second assay detects an agent-biased change in the PTEN pathway; (f) contacting the second assay system with the test agent of (b) under conditions whereby, but for the presence of the test agent, the system provides a reference

activity; and (g) detecting an agent-biased activity of the second assay system, wherein a difference between the agent-biased activity and the reference activity of the second assay system confirms the test agent as a candidate PTEN pathway modulating agent. Support for the amendment is found throughout the specification and in original claim 16.

New claim 30 recite "[t]he method of claim 8, wherein the nucleic acid modulator is a dsRNA or an siRNA. Support for the amendment is found throughout the specification and particularly at pages 18-19 and 40-41.

The claim amendments are made solely in an effort to advance prosecution and are made without prejudice, without intent to acquiesce in any rejection of record, and without intent to abandon any previously claimed subject matter. No new matter has been added by way of these amendments.

35 USC 112, First Paragraph Rejections

Claims 1, 8-12, 17-19, and 26-28 remain rejected under 35 USC 112, first paragraph as failing to comply with the written description requirement. Applicants respectfully traverse the rejections.

The Office alleges that the claims fail to comply with the written description requirement because they recite "'fragments or derivatives with kinase activity' which can encompass any kinase with any sequence or function". Office Action, page 3. However, the claims have been amended to delete reference to a SNF1LK derivative and also to clarify that the functionally active SNF1LK fragment encodes a polypeptide comprising amino acid residues 27 – 278 of SNF1LK and has kinase activity. Thus, the claims no longer encompass the use of "any fragment or derivative which encodes a polypeptide with kinase activity" and instead is limited to those fragments encoding a polypeptide comprising amino acids 27-278 of SNF1LK and having functional kinase activity.

As taught in the specification and known in the art, amino acid residues 27-278 represent the functional domain of the SNF1LK protein (i.e., the kinase domain), which is described in the specification at pages 5 and 36 (SEQ ID NO: 15 is the polypeptide encoded by SEQ ID NO: 5). Thus, the specification describes the invention in such clear, concise, exact terms that a skilled artisan would have recognized that Applicants were in possession of the claimed invention.

Accordingly, Applicants respectfully request withdrawal of the 35 USC 103(a) rejections.

35 USC 103(a) Rejections

Claims 1, 8-12, 17-18, 26 and 28 are rejected under 35 USC 103(a) as being unpatentable over Drewes et al. (Cell, 89:297-308 (1997) in view of Martinez et al., PNAS, 99: 14849-14854 (2002) further in view of Arora et al. Drug Metab and Dispos, 30: 757-762. Applicants respectfully traverse the rejections.

The Office alleges that Drewes et al. teaches that MARK phosphorylates MAP2 and MAP4 and causes their dissociation from microtubules, which results in the disruption of microtubule arrays leading to morphological changes of cells. The Office also alleges that Drewes et al. teaches an assay wherein CHO cells were transformed with a vector comprising a polynucleotide that encodes MARK1 or MARK2 and a vector expressing MAP2c (alleged test agent) or in the presence or absence of taxotere (an agent that stabilizes microtubules). The Office argues that differential phenotypes were seen in both of the assays and concludes that it would have been obvious to an ordinary person skilled in the art to search for an agent that interferes with the activation of MARK in view of preventing its ability to phosphorylate MAP kinases.

Applicants submit that Drewes et al. fails to render obvious the claimed invention.

As stated in Chapter 706.02(j) of the MPEP:

> "To support the conclusion that the claimed invention is directed to obvious subject matter, either the references must expressly or impliedly suggest the claimed invention or the examiner must present a convincing line of reasoning as to why the artisan would have found the claimed invention to have been obvious in light of the teachings of the references." *Ex parte Clapp*, 227 USPQ 972, 973 (Bd. Pat. App. & Inter. 1985).

In view of the Restriction Requirement mailed on March 23, 2009, Applicants amended the claims to recite the use of a single gene in the claimed assay, which gene is SNF1LK (SEQ ID NO: 5). Drewes et al. makes no mention of SNF1LK and therefore fails to provide any teaching with respect to SNF1LK, and, in particular, fails to provide any teaching or suggestion of an association between SNF1LK and the PTEN pathway. Therefore, Drewes et al. fails to expressly or impliedly suggest the elements of the claimed invention.

Further, the disclosure of Martinez et al. fails to cure the deficiencies of Drewes et al. The disclosure in Martinez et al. is limited to a general teaching of siRNA and fails to teach the use of siRNA to degrade SNF1LK nucleic acid in cells. Likewise, Arora et al. fails to cure the deficiencies of Drewes et al. Arora et al. teach that antisense phosphorodiamidate morpholino oligomers (PMO) inhibit targeted gene expression by preventing ribosomal assembly, but fails to teach using a SNF1LK-specific PMO for inhibition of SNF1LK expression in cells.

In the absence of any teaching whatsoever relating to the function of SNF1LK, the modulation of SNF1LK, or the association of SNF1LK and the PTEN pathway found in the disclosures of Drewes et al, Martinez et al, and Arora et al, these references, alone or in combination, fail to teach or suggest the claimed assays and thus fail to render obvious the present invention. Accordingly, Applicants respectfully request withdrawal of the 35 USC 103(a) rejections.

Conclusion

The applicants respectfully submit that all conditions of patentability are met in the pending claims as amended. Allowance of the claims is thereby respectfully solicited. If there are any questions or comments regarding this application, the Examiner is encouraged to contact the undersigned in order to expedite prosecution.

Respectfully submitted,

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/Anita J. Terpstra/

Anita J. Terpstra

Registration No. 47,132

McDonnell, Boehnen, Hulbert & Berghoff LLP
300 S. Wacker Drive
Chicago, IL 60606
(312) 913-0001